

THE INHERITANCE OF FIELD REACTION TO  
WHEAT SOIL-BORNE MOSAIC IN SEVEN  
WINTER WHEAT CULTIVARS

by

RASHIED S. MODAWI  
B.Sc., University of Khartoum, Sudan, 1976

A MASTER'S THESIS

submitted in partial fulfillment of the

requirements for the degree

MASTER OF SCIENCE

Genetics

KANSAS STATE UNIVERSITY  
Manhattan, Kansas  
1979

Approved by

  
Major Professor

Spec Coll.  
LD  
2668  
T4  
1979  
M62  
C.2

TABLE OF CONTENTS

	Page
LIST OF TABLES. . . . .	iii
INTRODUCTION. . . . .	1
REVIEW OF THE LITERATURE. . . . .	1
MATERIALS AND METHODS . . . . .	3
RESULTS AND DISCUSSION. . . . .	7
Analysis of the F <sub>1</sub> and F <sub>2</sub> Populations for the Crosses Gage/KS73256, Eagle/KS73256, Centurk/KS73256, Centurk/Gage and their Reciprocals. . . . .	7
Crosses Involving Centurk. . . . .	10
Gage/Shawnee, Shawnee/Gage . . . . .	11
Eagle/Shawnee, Shawnee/Eagle . . . . .	11
Eagle/Gage, Gage/Eagle . . . . .	11
Crosses of the Resistant X Resistant Cultivars . . . . .	16
Oasis X Gage - Gage X Oasis. . . . .	16
Oasis X Eagle - Eagle X Oasis. . . . .	19
Gage X KS73148, KS73148 X Gage . . . . .	19
Eagle X KS73148, KS73148 X Eagle . . . . .	19
SUMMARY . . . . .	20
ACKNOWLEDGMENT. . . . .	22
REFERENCES. . . . .	23

## LIST OF TABLES

Table	Page
1. Wheat soil-borne mosaic reaction of the parents used in the complete diallel cross with some distinguishing head characters - Manhattan, Kansas, 1976-1979. . . . .	4
2. The field reaction of seven parents and $F_1$ plants from a complete diallel crosses to wheat soil-borne mosaic in Manhattan, Kansas, Spring 1976. . . . .	8
3. The field reaction of $F_1$ plants, $F_2$ populations and the $F_3$ lines to WSBM in Manhattan, Kansas, 1976-1979 . . . . .	9
4. The field reaction of $F_1$ plants, $F_2$ populations and $F_3$ lines from Centurk crosses to WSBM in Manhattan, and Hesston, Kansas, 1976-1979 . . . . .	12
5. The observed and expected ratios of WSBM-diseased and undiseased $F_3$ lines from some crosses of resistant X susceptible cultivars in Manhattan, Kansas, 1976-1979 . . . . .	13
6. Field reaction of $F_1$ plants and $F_2$ populations from resistant X resistant crosses to WSBM in Manhattan, Kansas, 1976-1979 . . . . .	17
7. The observed and expected ratios of WSBM-diseased and undiseased $F_3$ lines from some crosses of resistant X susceptible cultivars in Manhattan, Kansas, 1976-1979. . . . .	18

## INTRODUCTION

Wheat soil-borne mosaic virus, the cause of wheat soil-borne mosaic disease is considered a serious threat to wheat production in Kansas and other wheat producing states in the U.S. Resistant cultivars are recognized as the most practical control for the disease. Breeding for resistance will be easier with a thorough understanding of the genetic system that gives field resistance. This study was conducted to add more information on the mode of inheritance of reaction to the disease in some common wheat cultivars.

## REVIEW OF LITERATURE

Wheat soil-borne mosaic is a disease of wheat Triticum aestivum (L.) em. Thell, caused by the soil-borne virus (WSBMV). It is transmitted by the zoospores of the fungus Polymyxa graminis (Estes and Brakke, 1966) which lives on the roots of wheat, barley and rye and other grasses. The virus consists of two particle types, each 26 nm in diameter but different lengths, 160 and 300 nm (Herbert and Coleman, 1955). Both subunits of the virus have to be present for infection. McKinney (1925) described two symptom expressions of the disease, the mosaic-rosette which was observed in a few cultivars in Illinois and some eastern states and the mosaic expression which is the most common. He correlated the two phases to two different strains of the virus, the green and the yellow strain respectively. Based on the symptoms, Sili (1958) concluded that only the yellow strain is found in Kansas and the Great Plains.

The first epiphytotic to occur in Kansas was in 1949-1951 (Fellows et al., 1953). In 1969 the disease was reported to have spread into the main wheat-producing fields of south central Kansas (Campbell et al., 1975). Loss in

yield due to the disease increased from an average of 17% in 1957 to 46% in 1973 (Sill 1958 and Campbell et al., 1975 Nykaza, et al., 1979). Similar high losses in wheat were reported in Florida and Nebraska (Kucharek and Walker, 1974, and Palmer and Brakke, 1975).

In 1958 Sill stated that the development of the individual plant symptoms appear to depend on the level of soil infestation, the environment and susceptibility of the cultivar. Different workers tried to eliminate the disease by decreasing the level of soil infestation (McKinney, 1923, Pacumbaba, 1966 and Kucharek et al., 1974). Although a considerable decrease in disease incidence was obtained by soil fumigation it was agreed that it is not a practical method for control. The fumigants also may alter some beneficial biological factors in the soil. McKinney (1923) indicated that different cropping methods failed to control the disease. Extremely late fall seeding reduced symptoms, but seeding must be late enough so that the plants do not emerge until the following spring. In Kansas the date of planting had no effect on the incidence of the disease <sup>but</sup> by the environment considerably affected the expression of the disease (Sill, 1958 and Nykaza et al., 1979).

Resistance to the disease has been observed in the field (Roane et al., 1954). McKinney (1925) was able to select resistant plants from fields of a susceptible cultivar. McKinney (1925) also showed resistance to be under genetic control and it was possible to breed resistant cultivars. As the disease was found to be an important factor in wheat production in different states, breeding for resistance to the disease was placed among the priorities of different research stations (Roane et al., 1954, Koehler et al., 1952, Sill et al., 1960, Kucharek and Walker, 1974, Campbell et al., 1975 and Nykaza et al., 1979).

The control of wheat soil-borne mosaic through the development of resistant cultivars was recognized as the most practical control for the disease, nevertheless work on the genetics of the disease was not comparable to the significant role the disease plays in wheat production. Miyake (1939) in Japan evaluated the  $F_1$  and  $F_2$  progenies from a series of crosses of wheat cultivars. He reported resistance to both the yellow and the green mosaic to be due to a single dominant gene. Nakagawa et al. (1959) evaluated the reaction of the  $F_2$  generation in crosses of susceptible and resistant cultivars to the green and yellow mosaic separately in Japan. They found that the same system operates for both diseases, that is; three loci with multiple alleles, the two loci H and M determining susceptibility and one locus, the A locus inhibits the H locus. A different system was found to operate in the United States wheats. Shaalan et al. (1966) studied the reaction of  $F_2$  progenies of two crosses to the yellow mosaic. They found resistance to be controlled by two factors, a partially dominant gene for resistant and a modifying gene. This could be due to difference in both the virus strains involved or the genotypes of wheat used in the U.S. and Japan. Dubey et al. (1970) studied the reaction of  $F_1$ ,  $F_2$ ,  $F_3$  and backcrossed  $F_2$  families of five-parent incomplete diallel cross to the mosaic and mosaic-rosette disease in Illinois. They found resistance to be monogenic dominant over susceptibility to both the mosaic and mosaic-rosette phases of the disease. They also found susceptibility to the mosaic to be dominant over susceptibility to the mosaic rosette.

#### MATERIALS AND METHODS

Seven winter wheat cultivars with different levels of field reaction to wheat soil-borne mosaic (WSBM) were used for the study (Table 1). Six of the



Table 1. Wheat soil-borne mosaic reaction of the parents used in the complete diallel cross with some distinguishing head characters - Manhattan, Kansas 1976-1979.

Cultivar	C I No. or Selection No.	Reaction to WSBM*	Grain Color	Glume Color	Bearded or Awnless
Gage	CI 13532	MS	Red	White	Bearded
Shawnee	CI 14157	R	Red	Red	Bearded
Eagle	CI 15068	S	Red	White	Bearded
Centurk	CI 15075	MR	Red	White	Bearded
Oasis	CI 15929	R	Red	White	Awnless
CIMMYT/Scout	KS73256	R	White	Red	Bearded
CIMMYT/Scout	KS73148	R	Red	White	Bearded

\*MS = moderately susceptible, S = susceptible, MR = moderately resistant, R = resistant.

cultivars were hard red winter wheats and the other was a soft winter wheat. Gage and Eagle were used as the susceptible parents. Gage is less susceptible than Eagle and often classified as moderately susceptible. Centurk is variable in its reaction to WSBM and generally classified as moderately resistant. Shawnee, KS73256, and KS73148 are resistant. Oasis which was the only soft winter wheat was resistant but has some yellowing on the leaves which could be mistaken for the mosaic symptoms. Besides the known reaction to WSBM, two other head characters were used as markers for the crosses, as given in Table 1.

The reaction of the parents and the  $F_1$  plants were classified according to the leaf symptoms and growth habit into the following classes:

Resistant (R): no mottling on the leaves, no stunting.

Moderately Resistant (MR): very slight mottling with no stunting.

Moderately Susceptible (MS): mottling obvious with some stunting.

Susceptible (S): severe mottling on the leaves and severe stunting.

The seven cultivars were crossed in all possible combinations including the reciprocals. The 42 crosses were made in the greenhouse. About 10 seeds from each cross were seeded in the fall of 1975 in naturally infested field, but due to poor seed germination in some crosses there were variable numbers of  $F_1$  plants which reached maturity.

The  $F_2$  populations were seeded in the fall of 1977 in infested soils at Hesston (south central Kansas) and at the Hartner field at Ashland area of Manhattan, Kansas together with the seven parents. They were also space-seeded in 8-foot rows at the Agronomy Farm Unit #3 at Ashland. Plants from the latter location were harvested individually and threshed separately. From each cross an average of 70 plants were obtained.



The  $F_2$  populations were classified into three classes: the resistant (R) and moderately resistant were classified as resistant; the susceptibles and moderately susceptibles were classified as susceptible; and the third class was the segregating populations.

In the fall of 1978 a progeny test for the  $F_2$  populations was made. A random sample of 25 seeds from each single plant progeny were seeded as the  $F_3$  lines in 3-foot rows. The parental checks were seeded every 20th and 21st rows. The  $F_3$  lines were seeded in two locations at Ashland, one at Unit #3 of the Agronomy Farm and the other at the Hartner field. In the latter locations the seed bed was very poor, poor germination, late emergence and low infection caused abandonment of this seeding. At Unit #3 the field germination was fairly uniform and the infection level was reasonable to produce symptoms on susceptible plants. Only the  $F_3$  data from Unit #3 were used for analysis. In this plot the environment favored the expression of the symptoms as early as the first week of March. The leaf symptoms were seen till the first week of May.

Readings for WSBM infection were taken when the wheat plant had resumed spring growth, in early April 1979 and checked two more times in mid and late April. The color of the glumes and the presence or absence of the awns in the  $F_2$  populations were used to check the populations.

The statistical test used was the  $\chi^2$  test as suggested by Harris (1912). A modification for the test recommended by Fisher (1954), when the number of individuals in each class was less than 5, required the combining of the susceptible class and the segregating class into one class, that is, the diseased class. The modification served a two-fold function. First it helped meet Fisher's recommendation for the minimum number of individuals in each

class and second it eliminated the possible discrepancy which arises when there is an escape from the virus or the virus level is not uniform in the soil. Under such conditions it would be difficult to differentiate between the truly segregating populations and those which were susceptible but some of the plants were not infected. So the  $X^2$  test was used for two classes, the resistant class and the diseased class.

## RESULTS AND DISCUSSION

### Analysis of the $F_1$ and $F_2$ Populations for the Crosses Gage/KS73256, Eagle/KS73256, Centurk/KS73256, Centurk/Gage and their Reciprocals

From the reaction of the  $F_1$  plant to WSBM (Table 2) it was seen that resistance to the disease was dominant over susceptibility. The resistant X resistant and resistant X susceptible gave resistant  $F_1$  plants. The susceptible X susceptible cross gave susceptible  $F_1$  plants.

Four of the combinations gave reciprocal difference in the  $F_1$ s. Those are listed in Table 3 with their  $F_1$  and  $F_2$  reaction and the number of plants in each class of the  $F_2$  population.

Such differences between reciprocals suggests the involvement of non-chromosomal factors in the inheritance of reaction to the virus. Gage, Eagle and Centurk produced susceptible  $F_1$  hybrids when they were the female parent, the male parent being KS73256 and Gage.

The fact that only those combinations produced the reciprocal differences in the  $F_1$  would indicate that the male parent genome is also involved.

Generally reciprocal difference in the  $F_1$  indicate either of two possibilities:

1. An environmentally induced factor left over in the female egg which stimulated an other nucleus or cytoplasmic factor for susceptibility in the

Table 2. The field reaction of seven parents and F<sub>1</sub> plants from complete diallel crosses to WSBM in Manhattan, Kansas, in spring of 1976.

Cross	Reac- tion*	# of plants	Cross	Reac- tion*	# of plants	Cross	Reac- tion*	# of plants
Reaction* of F <sub>1</sub> Plants								
Shawnee/Gage	R	3	Gage/Centurk	MR	6	Eagle/Oasis	MR	2
Gage/Shawnee	R	8	Centurk/Gage	S	5	Oasis/Eagle	MR	1
Shawnee/Eagle	R	3	Gage/KS73256	S	4	Centurk/KS73256	S	2
Eagle/Shawnee	MR	3	KS73256/Gage	R	9	KS73256/Centurk	MR	5
Shawnee/Centurk	R	7	Gage/KS73148	R	6	Centurk/KS73148	R	6
Centurk/Shawnee	MR	5	KS73148/Gage	R	4	KS73148/Centurk	R	8
Shawnee/KS73256	R	9	Gage/Oasis	R	4	Centurk/Oasis	MR	6
KS73256/Shawnee	MR	8	Oasis/Gage	MR	5	Oasis/Centurk	MR	4
Shawnee/KS73148	R	4	Eagle/Centurk	S	5	KS73256/KS73148	R	3
KS73148/Shawnee	R	7	Centurk/Eagle	S	4	KS73148/KS73256	MR	2
Shawnee/Oasis	R	6	Eagle/KS73256	S	1	KS73256/Oasis	MR	2
Oasis/Shawnee	R	8	KS73256/Eagle	R	8	Oasis/KS73256	R	7
Gage/Eagle	S	6	Eagle/KS73148	R	5	KS73148/Oasis	R	7
Eagle/Gage	S	6	KS73148/Eagle	R	3	Oasis/KS73148	R	5
Reaction* of Parents								
Shawnee (R)			Centurk (MR)			KS73148 (R)		
Gage (MS)			KS73256 (R)			Oasis (R)		
Eagle (S)								

\*R = resistant, MR = moderately resistant, S = susceptible, MS = moderately susceptible.

Table 3. The field reaction of  $F_1$  plants,  $F_2$  populations and the  $F_3$  lines to WSBM in Manhattan, Kansas, 1976-1979.

Cross	$F_1$	$F_2$ Population	$F_3$ Lines
	Reaction*	Reaction*	R : Seg: S
Gage/KS73256	S	R & MS	26 : 32 : 6
KS73256/Gage	R	R & MS	31 : 26 : 14
Eagle/KS73256	S	R & MS	14 : 34 : 22
KS73256/Eagle	R	R & MS	46 : 25 : 2
Centurk/KS73256	S	R & MS	21 : 20 : 3
KS73256/Centurk	R	R & MS	11 : 20 : 10
Centurk/Gage	S	R & S	2 : 11 : 20
Gage/Centurk	MR	R & MS	30 : 11 : 15

\*R = resistant, MR = moderately resistant, S = susceptible, MS = moderately susceptible, Seg = Segregating

$F_1$ . This is what is known as maternal effect. It is not expected to continue its effect in the next generation since the original signal was not inherited.

2. A cytoplasmic factor for susceptibility (a plasmon) which is self-propagating. So it will continue to show in the next generations as long as the same cytoplasm is being used. The progeny of  $F_1$  which is susceptible will be all susceptible and so will be the next generations. However if this cytoplasmic factor is under the control of some chromosomal factor(s) the  $F_2$  population will tend to segregate in the normal Mendelian ratios.

From Table 3 it seems that Gage, Eagle and Centurk cytoplasms has a cytoplasmic factor for susceptibility which was dominant over the resistance of KS73256. It is also possible that those cytoplasmic factors were induced by nuclear factors in KS73256 in the above 3 cultivars and also by Gage in Centurk. The nuclear control over the cytoplasmic factors can be seen in the  $F_2$  reaction of those crosses. It shows the segregation ratios which would be expected if there were two or more recessive nuclear factors which induce susceptibility of the cytoplasm.

However, the evidence available from this study will not exclusively support the above hypothesis and two or more cycles of reciprocal backcrosses are recommended to confirm the presence or absence of cytoplasmic factors.

#### Crosses Involving Centurk

It is believed that the Centurk population used in this study was a mixture of more than one genotype. The parental checks were seeded together with the  $F_2$  lines. They were also the progeny of a single plant. Centurk lines showed variable reaction to the virus. Some lines were as resistant as Shawnee, others were as susceptible as Gage. There was such an inconsistency and absence of definite pattern in Centurk reaction that in the cross



with Eagle more than 90% of the  $F_2$  population was susceptible and in the cross with Oasis only one susceptible line from a total of 123  $F_3$  lines was observed (Table 4). In the first case it seems as if Centurk has the same genotype as Gage, in the second case it has the genotype of Shawnee or KS73256. Still in the cross with Shawnee and the cross with Gage the  $F_2$  segregation ratio indicate a difference between those two genotypes and Centurk. It was also segregating for ratio close to 7:8:1 in the crosses with KS73256 and KS73148.

Gage/Shawnee, Shawnee/Gage -- Table 5

The  $F_1$  and the  $F_2$  segregation of this cross showed no difference between the reciprocals. Therefore the information was combined into one set of data. Accordingly the segregation ratio will be 60 R: 43 Seg: 23 S. If we also combine the segregating class then the susceptible class data will give a very close fit to the 7:9, which is 7:8:1, ratio indicating a difference of two genes.

Eagle/Shawnee, Shawnee/Eagle -- Table 5

Treating the data in the same manner as the above cross, this will also give a close fit to the 7:9 ratio with a probability between .10 - .05.

Eagle/Gage, Gage/Eagle -- Table 5

In both crosses the  $F_1$  was susceptible. To this point Gage and Eagle would be considered to have the same genotype since with Shawnee they gave the same  $F_2$  segregation ratio. But the  $F_2$  reaction of Gage/Eagle and Eagle/Gage also indicated a two gene difference between the two cultivars. The fact that the two cultivars were susceptible, their  $F_1$  was susceptible, and some  $F_3$  lines were breeding true for resistance, would indicate that the



Table 4. The field reaction of  $F_1$  plants,  $F_2$  populations and  $F_3$  lines from Centurk crosses to WSBM<sup>1</sup> in Manhattan, and Hesston, Kansas, 1976-1979.

Cross	$F_1$ Reaction	$F_2$ Population Reaction*	$F_3$ Lines R : Seg: S
Eagle/Centurk	S	R - <u>S</u>	1 : 30 : 33
Centurk/Eagle	S	R - <u>S</u>	9 : 39 : 19
Centurk/KS73148	R	<u>R</u> - S	28 : 25 : 10
KS73148/Centurk	R	<u>R</u> - S	22 : 29 : 10
Centurk/Oasis	MR	R	46 : 12 : 0
Oasis/Centurk	MR	R	59 : 5 : 1
Centurk/Shawnee	MR	<u>R</u> - S	38 : 31 : 3
Shawnee/Centurk	R	<u>R</u> - S	34 : 19 : 11

\*Underlined response indicates the most frequent class.

Table 5. The observed and expected ratios of WSBM-infected and healthy  $F_3$  lines from some crosses of resistant X susceptible cultivars in Manhattan, Kansas, 1976-1979.

Cross	$F_1^*$ Reaction	Observed $F_3$ lines R : Seg: S	Observed H : I	Expected Ratio	$\chi^2$	P**
Gage/Shawnee	R	34 : 19 : 11	34 : 30	7:9		.5-.25
Shawnee/Gage	R	26 : 24 : 12	26 : 36	7:9	.80	
Gage/Eagle	S	3 : 17 : 32	3 : 49	1:15		
Eagle/Gage	S	2 : 32 : 36	2 : 68	1:15	.95	.5-.25
Eagle/Shawnee	R	21 : 24 : 17	1 : 41	7:9		
Shawnee/Eagle	R	22 : 28 : 9	22 : 37	7:9	3.35	.1-.05

\*R = resistant, Seg. + segregating lines, S = susceptible, I = infected, H = health.

\*\*Probability of a higher  $\chi^2$  value (Snedecor and Cochran, 1967).

resistant lines and segregation was brought by genetic recombination either complementary gene action or modifying factor(s). The observed ratio of resistant: diseased lines in the  $F_3$  gave a high probability of fit to 1:15 segregating ratio which will fit in both hypothesis, modifying factor or complementary genes. Here it is suggested that a modifying gene is involved since it was seen to better explain the reaction of Gage, Eagle, and their cross with each other and with the other resistant cultivars.

From the above three combinations it is suggested that Gage is homozygous dominant at two loci, the B locus which conditions resistance when it is dominant, the other locus is C which inhibits the action of B. Thus the moderately susceptible Gage will have the genotype--BBCC. Eagle is suggested to have the genotype bbcc which is susceptible.

According to this hypothesis the breeding behavior of the Gage/Eagle cross can be followed as below:

BBCC X bbcc		
$F_1$	Bb Cc	Susceptible - segregate
$F_2$	1 BB CC *	breed true susceptible
	2 BB Cc *	segregate in 1 R : 3 S
	1 BB cc	breed true resistant
	2 Bb CC	breed true susceptible
	4 Bb Cc	segregate in 1 R : 15 S
	2 Bb cc	segregate in 3 R : 1 s
	1 bb CC	breed true susceptible
	2 bb Cc	breed true susceptible
	1 bb cc *	breed true susceptible

\* parental genotypes

Going back to the cross of Eagle and Shawnee, the two gene difference is explained as Shawnee being homozygous dominant at the two loci A and B each of them is capable of conditioning resistance when it is dominant. So in the cross of the resistant Shawnee having the genotype - AA BB cc - with the susceptible Eagle having the genotype - aa bb cc - the F<sub>2</sub> will segregate as follows:

AA BB cc X aa bb cc		
F <sub>1</sub>	Aa Bb cc	resistant - segregate
F <sub>2</sub>	1 AA BB cc *	breed true resistant
	2 AA Bb cc	breed true resistant
	1 AA bb cc	breed true resistant
	2 Aa BB cc	breed true resistant
	4 Aa Bb cc	segregate 15 R : 1 S
	2 Aa bb cc	segregate 3 R : 1 S
	1 aa BB cc	breed true resistant
	2 aa Bb cc	segregate 3 R : 1 S
	1 aa bb cc *	breed true susceptible

\* parental genotypes

In the cross of Shawnee/Gage the F<sub>2</sub> segregation will be as follows:

AA BB cc X aa BB CC		
F <sub>1</sub>	Aa BB Cc	resistant
F <sub>2</sub>	1 AA BB CC	breed true resistant
	2 AA BB Cc	breed true resistant
	1 AA BB cc *	breed true resistant
	2 Aa BB CC	segregate 3 R : 1 S
	4 Aa BB Cc	segregate 13 R : 3 S

2 Aa BB cc	breed true resistant
1 aa BB CC *	segregate 1 R : 3 S
2 aa BB Cc	segregate 1 R : 3 S
1 aa BB cc	breed true resistant

\* parental genotypes

#### Crosses of the Resistant X Resistant Cultivars -- Table 6

Crosses of the resistant X resistant cultivars always gave resistant  $F_1$  and continued to breed true for resistance in the  $F_2$  populations and their progeny. Occasionally symptoms were observed on some plants of those lines but only less than 1% of the population showed the slight symptoms. The same kind of symptoms were also generally seen in the resistant cultivars like Shawnee and Oasis. It is possible that such reaction was due to outcrossing or seed mixtures.

Those crosses were: Shawnee X KS73256, Shawnee X KS73148, Shawnee X Oasis, KS73256 X Oasis, KS73256 X KS73148, KS73256 X Oasis, KS73148 X Oasis and their reciprocals (Table 6). The breeding behavior of those crosses would indicate that the cultivars involved all possessed at least one dominant gene for resistance in common.

#### Oasis X Gage - Gage X Oasis -- Table 7

Table 7 shows the crosses of Oasis X Gage, Gage X Oasis. The  $F_2$  segregation appeared to be like that of Shawnee X Gage, Gage X Shawnee. Test of the hypothesis for 7 resistant: 9 infected gave a  $\chi^2$  value - 0.63 with 0.5 - 0.25 probability of a higher  $\chi^2$  value. By comparing the three crosses, Shawnee X Gage, Shawnee X Oasis and Oasis X Gage, it could be concluded with high probability that Oasis has the same genotype as Shawnee, that is, AA BB cc.

Table 6. Field reaction of  $F_1$  plants and  $F_2$  populations from resistant X resistant crosses to WSEM in Manhattan, Kansas, 1976-1979.

Cross	$F_1$	$F_2$	Cross	$F_1$	$F_2$
Shawnee/KS73256	R	all R	KS73256/KS73148	R	all R
KS73256/Shawnee	MR	all R	KS73148/KS73256	MR	all R
Shawnee/KS73148	R	all R*	KS73256/Oasis	MR	all R
KS73148/Shawnee	R	all R*	Oasis/KS73256	R	all R
Shawnee/Oasis	R	all R*	KS73148/Oasis	R	all R
Oasis/Shawnee	R	all R*	Oasis/KS73148	R	all R

\*Had 4 lines out of 80  $F_3$  lines segregating.



Table 7. The observed and expected ratios of WSBM-infected and healthy  $F_3$  lines from some crosses of resistant X susceptible cultivars in Manhattan, Kansas, 1976-1979.

Cross	$F_1$ Reaction*	Observed $F_3$ Lines R : Seg: S	Observed H : D	Expected I : H	$X^2$	P**
Oasis/Gage	MR	42 : 34 : 7	42 : 41	7: 9	.63	.5-.25
Gage/Oasis	R	34 : 36 : 7	34 : 43	7: 9		
Oasis/Eagle	MR	18 : 30 : 11	18 : 41	1: 3	1.6	.25-.10
Eagle/Oasis	MR	18 : 30 : 13	18 : 43	1: 3		
Gage/KS73148	R	49 : 24 : 3	49 : 27	37: 27	1.6	.25-.10
KS73148/Gage	R	34 : 25 : 15	34 : 40	37: 27	.13	.75-.50
Eagle/KS73148	R	11 : 28 : 30	11 : 58	1: 3	2.23	.25-.10
KS73148/Eagle	R	15 : 22 : 28	15 : 50	1: 3		

\*R = resistant, Seg. = segregating lines, S = susceptible, MR = moderately susceptible, H = healthy, I = infected.

\*\*Probability of a higher  $X^2$  value. Snedecor and Cochran (1967).

Oasis X Eagle - Eagle X Oasis -- Table 7

When recording the  $F_2$  populations reaction, it was suspected that Oasis X Eagle was not the proper cross, although there was segregation for the awnless character of Oasis. Such decision depended only on the WSBM reaction. The whole  $F_2$  population was resistant. It is possible that the resistance of the  $F_2$  population was not due to the breeding behavior of the hybrid only, rather it was caused by the absence of virus inoculum from that part of the field. In fact when the progeny of the same population was seeded as  $F_3$  lines it segregated for resistant as well as susceptible lines. It is tempting to consider Eagle X Oasis and Oasis X Eagle were actually crosses. However when testing the  $F_3$  lines for the segregation ratio of 7:8:1 that is, 7 resistant : 9 infected, the  $\chi^2$  test gave a low probability of fit to the hypothesis. Instead it gave a close fit to the 1:2:1 segregation ratio with a probability of 0.50 - 0.25. If it was truly segregating for that ratio then the Oasis population used in this cross will have the genotype AA bb cc which is different than the genotype of Oasis used with Gage.

Gage X KS73148, KS73148 X Gage -- Table 7

The  $F_1$  of this cross was resistant. The  $F_2$  population seemed to segregate in the ratio 37:26:1. If so then the  $F_1$  was heterozygous for three loci. Since Gage has the genotype aa BB CC, then it is possible the KS73148 has one locus homozygous dominant, that is, AA bb cc.  $\chi^2$  test gave a good agreement with the hypothesis.

Eagle X KS73148, KS73148 X Eagle -- Table 7

From the above hypothesis about the genotype of KS73148 it is expected that the  $F_2$  population of this cross will segregate in the 1:2:1 ratio.

Although there seemed to be an excess of susceptible lines and less resistant lines in the  $F_3$  but when combining the segregating lines and the susceptible lines a  $\chi^2$  value of 2.23, with probability between 0.25 and 0.10 was obtained indicating a good fit to the hypothesis.

#### SUMMARY

The field reaction to wheat soil-borne mosaic was studied in the  $F_1$  and  $F_3$  generations of a seven-parent complete diallel cross. Under natural conditions of field infestation the plants were classified into resistant and susceptible according to the foliar symptoms observed after the wheat plants resumed the spring growth. Resistance was found to be dominant over susceptibility. This was evident in the  $F_1$  of most of the susceptible X resistant crosses. However, some of those crosses gave reciprocal differences in the  $F_1$ s, Table 3. This leads to the suggestion that Centurk, Gage and Eagle might possess some cytoplasmic factor(s) for susceptibility which was stimulated only by some nuclear factor(s) in KS73256.

Resistant X resistant crosses always gave resistant progeny indicating that the resistant cultivars has at least one locus conditioning resistance in common.

The resistant X susceptible crosses gave a resistant  $F_1$ , the  $F_2$  segregated in a simple Mendelian fashion.

From the analysis of the  $F_3$  lines of all the crosses it is suggested that the reaction of the wheat cultivars used in this study to WSBM is controlled by three loci. A and B conditioning resistance with complete dominance of resistance over susceptibility, and the C locus which produce an inhibitory effect on the B gene.

According to this system the behavior of six of the seven cultivar can be viewed as the expression of the following genotypes.

Eagle (susceptible) - aabbcc -

Gage (moderately susceptible) - aaBBCC -

Shawnee (Resistant) - AABBBcc -

KS73256 (Resistant) - AABBBcc -

KS73148 (Resistant) - AAbbccc -

Oasis (Resistant) - AABBBcc - or AAbbccc -

The irregular behavior of Centurk suggests that the Centurk population used was not homogenous. It contained some of the above genotypes in different proportions.

Although it has been suggested that there may be some difference in the wheat cultivars and the virus strains in the United States and Japan, the data obtained in the research reported here suggests there is a close agreement with the Japanese work (Nakagawa, et al., 1959).

## ACKNOWLEDGEMENT

The author would like to express his deep gratitude to Professor Elmer G. Heyne for suggesting the problem and for his valuable and patient guidance in the field and throughout the preparation of the manuscript. Thanks are also due to Dr. G. Liang and C. Niblett for serving as committee members, to the wheat breeding and genetics group and to the Department of Agronomy. Special thanks for the Government of Sudan for their support.

## REFERENCES

- Campbell, L. G., E. G. Heyne, D. M. Gronau, and C. Niblett. 1975. Effect of soil-borne wheat mosaic virus on wheat yield. Plant Disease Reporter. 59:472-476.
- Dubey, S. N., C. M. Brown, and L. L. Hooker. 1970. Inheritance of field reaction to soil-borne wheat mosaic virus. Crop Science. 10:93-95.
- Estes, A. P., and M. K. Brakke. 1966. Correlation of Polymyxa graminis with transmission of soil-borne wheat mosaic virus. Virology. 28:772-774.
- Fellows, H., W. H. Sill, Jr., and C. L. King. 1953. The 1952 epiphytotic of a soil-borne wheat mosaic in Kansas. Plant Disease Reporter. 37:287-289.
- Fisher, R. A. 1954. Statistical methods for research workers, 12th ed., Hafner New York pp. 78-113.
- Harris, J. A. 1912. A simple test of goodness of fit of Mendelian ratios. American Naturalist. 46:741-745.
- Herbert, T. T., and N. T. Coleman. 1955. Rod-shaped particles associated with soil-borne wheat mosaic. Phytopathology (Abstr.). 45:348.
- Koehler, B., W. M. Bever, and O. T. Bonnett. 1952. Soil-borne wheat mosaic. Illinois Agr. Exp. Sta. Bull. 556:567-599.
- Kucharek, T. A., and J. H. Walker. 1974. The presence and damage caused by soil-borne wheat mosaic virus in Florida. Plant Disease Reporter. 58:763-765.
- Kucharek, T. A., J. H. Walker, and R. D. Barnett. 1974. Effect of cultivar resistance and soil fumigation on soil-borne wheat mosaic virus in Florida. Plant Disease Reporter. 58:878-881.
- McKinney, H. H. 1923. Investigation of the rosette disease of wheat and its control. Journal of Agricultural Research. 23:771-800.
- McKinney, H. H. 1925. A mosaic disease of winter wheat and winter rye. U. S. Dept. Agr. Bull. 1361.
- Miyake, M. 1938. Mendelian inheritance of resistance against the virus disease in wheat strains. Japanese J. Genetics. 14:239-242. (Abstr. in Plant Breeding, Abstracts 1937-1939 Vol. VIII, p. 300).
- Nakagawa, M., Y. Soga, N. Okasima, A. Yoshioka, and D. Nisimata. 1958. Genetical studies on the wheat mosaic virus I. Genes affecting the inheritance of susceptibility to strains of green mosaic virus in



- varietal crosses of wheat. Japanese J. Breeding. 8:169-170. (Abstr. in Plant Breeding Abstracts 1959 Vol. 29, p. 739).
- Nakagawa, M., Y. Soga, S. Watanabe, H. Gocho, and K. Nishio. 1959. Genetical studies on the wheat mosaic virus II. Genes affecting the inheritance of susceptibility to strains of yellow mosaic virus in varietal crosses of wheat. Japanese J. Breeding. 9:118-120. (Abstr. in Plant Breeding Abstracts 1960 Vol. 30, p. 508).
- Nykaza, S. M., E. G. Heyne, and C. L. Niblett. 1979. The effect of wheat soil-borne mosaic on several plant characters of winter wheat. Plant Disease Reporter. July 1979 (in print).
- Pacumbaba, R. P. 1966. Transmission of soil-borne wheat mosaic virus. Masters thesis, Kansas State University, Kansas.
- Palmer, L. T., and M. K. Brakke. 1975. Yield reduction in winter wheat infected with soil-borne wheat mosaic virus. Plant Disease Reporter. 59:469-471.
- Roane, C. W., T. M. Starling, and H. H. McKinney. 1954. Observations on wheat mosaic in Virginia. Plant Disease Reporter. 38:14-18.
- Shaaalan, M. I., E. G. Heyne, and W. H. Sill, Jr. 1966. Breeding wheat for resistance to soil-borne wheat mosaic virus, wheat streak-mosaic virus, leaf rust, stem rust and bunt. Phytopathology. 56:664-668.
- Sill, W. H., Jr. 1958. A comparison of some characteristics of soil-borne wheat mosaic virus in the Great Plains and elsewhere. Plant Disease Reporter. 42:912-922.
- Sill, W. H., Jr., H. Fellows, and E. H. Heyne. 1960. Reaction of winter wheat to soil-borne wheat mosaic virus in Kansas. Kansas Agri. Exp. Sta. Tech. Bull. 112.
- Snedecor, G. W., and W. G. Cochran. 1967. Statistical methods 6th ed. The Iowa State University Press Ames, Iowa, U. S. 593p.

THE INHERITANCE OF FIELD REACTION TO  
WHEAT SOIL-BORNE MOSAIC IN SEVEN  
WINTER WHEAT CULTIVARS

by

RASHIED S. MODAWI  
B.Sc., University of Khartoum, Sudan, 1976

AN ABSTRACT OF A MASTER'S THESIS

submitted in partial fulfillment of the

requirements for the degree

MASTER OF SCIENCE

Genetics

KANSAS STATE UNIVERSITY  
Manhattan, Kansas  
1979

# ABSTRACT

The inheritance of field reaction to wheat soil-borne mosaic in winter wheat was studied in the  $F_1$ ,  $F_2$  and  $F_3$  generations of a seven parent complete diallel cross. The resistant parents were Shawnee, KS73148, KS73256 and Oasis. Centurk was moderately resistant, Gage moderately susceptible and Eagle was susceptible. Resistance was found to be dominant over susceptibility. The analysis of the  $F_3$  lines showed the field reaction to WSBM to be controlled by three loci; A and B, conditioning resistance and the third locus, C, inhibiting resistance of the B locus. The parents were suggested to have the genotypes: Shawnee and KS73256 = - AABbCc -, KS73148 = - AAbbCc -, Oasis = AABbCc - and/or - AAbbCc -, Gage = - aaBBCC - and Eagle = - aabbcc -. Centurk was believed to be a mixture of the above genotypes. It was also suggested that Centurk, Gage and Eagle possess some cytoplasmic factors for susceptibility. This was evident from the reciprocal differences in some  $F_1$  plants.